DEVELOPMENT OF EVENT-RELATED POTENTIALS (ERPS) IN INFANTS AND PRESCHOOL CHILDREN WITH AND WITHOUT FAMILIAL RISK FOR DYSLEXIA

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ABSTRACT

The maturation of auditory event-related potentials (ERPs) is complex. Different ERP components have different developmental time schedules regarding to amplitudes and latencies. Decreased amplitude of the negative deflection mismatch negativity (MMN) has been connected to the language learning deficits. In the present study 52 infants aged 6 months and the same subjects in childhood aged 6,5 years were studied by using an oddball paradigm. The purpose was to find out the developmental courses of the ERP peaks and the differences in the detection of temporal changes of speech between the groups of control and at-risk for developmental dyslexia. The standard stimulus was a pseudoword /ata/ and the temporally different deviant stimulus /atta/ was a longer version of the same pseudoword. It was found that latencies of the deflections $P_{i190/c110}$ and $N_{i320/c225}$ shortened with age. Amplitudes decreased for $P_{i190/c110}$ and increased for $N_{i320/c225}$ with age. $P_{i190/c110}$ was thought to be a precursor of an adult P1 and $N_{i320/c225}$ of an adult N2. In childhood there probably was some sign of N1. The groups differed at the both ages in their automatic detection of temporal changes of speech. This shows that individuals at-risk for developmental dyslexia have deficient perception of temporal features of speech. However, this deficit had somewhat eased from infancy to childhood.

TIIVISTELMÄ

Auditiivisten tilannesidonnaisten jännitteiden (ERP) kypsyminen on monimutkaista. Eri komponenteilla on erilaiset kehitykselliset aikataulut koskien amplitudeja ja latensseja. Poikkeavuus-negatiivisuus aallon (MMN) pienentyneen amplitudin on todettu olevan yhteydessä kielen oppimisvaikeuksiin. Tässä tutkimuksessa tutkittiin 52 vauvaa 6 kuukauden iässä ja samat yksilöt lapsuudessa 6,5 vuoden ikäisinä lapsuudessa käyttäen ns. oddball-koeasetelmaa. Tarkoituksena oli selvittää tilannesidonnaisten jännite-komponenttien kehitystä ja puheen temporaalisten erojen havaitsemiseroja kontrolliryhmässä ja kehityksellisen dysleksian riskiryhmässä. Standardiärsyke oli epäsana /ata/ ja temporaalisesti erilainen, poikkeava ärsyke /atta/ oli pidempi versio samasta epäsanasta. Piikkien $P_{i190/c110}$ ja $N_{i320/c225}$ latenssien todettiin lyhenevän iän myötä. Komponentin $P_{i190/c110}$ amplitudit pienenivät ja $N_{i320/c225}$ amplitudit kasvoivat iän myötä. $P_{i190/c110}$ ajateltiin olevan aikuisen P1 komponentin edeltäjä ja $N_{i320/c225}$ ajateltiin edustavan aikuisen N2:ta. Lapsuudessa näkyi ehkä merkki N1 aallosta. Ryhmät erosivat kummassakin iässä puheen temporaalisten erojen automaattisessa havaitsemissa. Tämä osoittaa, että kehityksellisen dysleksian riskissä olevilla yksilöillä on puheen temporaalisten piirteiden puutteellinen havaitsemiskyky. Tämä ongelma oli kuitenkin lieventynyt hieman lapsuudessa verrattuna vauva-ikään.

CONTENTS

1. INTRODUCTION

1.1 Developmental dyslexia

Developmental dyslexia is a learning disability that affects ability to learn to read during development in spite of adequate intelligence, opportunity to learn, adequate instruction and a supporting home environment (Critchley, 1970). According to Pennington (1995), dyslexia is a neurodevelopmental disorder and aggregates in families. The heritability of dyslexia has been studied in family and twin studies, segregation analyses and linkage analyses. It has been found that there may be a major gene effect, with reduced penetrance in females in hereditary of dyslexia. Evidence for the polygenic model has also been found. Dyslexia has been linked to the loci on chromosomes 6 and 15 (for a review see Schulte-Körne, 2001). Taipale et al. (2003) have proposed that *DYX1C1* in chromosome 15q21 should be regarded as a candidate gene for developmental dyslexia. In the review of Vellutino, Fletcher, Snowling, & Scanlon (2004), the heritability of dyslexia has been estimated to be 50 – 60 %.

Dyslexic individuals have problems in phonological coding i.e. encoding written language (graphemes) to representations of spoken language (phonemes). This may be due to a poor phonological awareness and poorly specified phonological representations (Ramus, 2003; Vellutino et al., 2004.) Dyslexic individuals have been reported to differ from normal readers by certain brain structures. The differences in the size of the left planum temporale have been thought to be related to phonological coding deficits (for a review, see Leppänen, Choudhury, Benasich, & Lyytinen, 2004). Problems in phonological coding and awareness leads to assumption, that dyslexic individuals have problems in speech perception and it gets support, for example, from the study of Mody, Studdert-Kennedy, & Brady (1997) and Lyytinen et al. (in press), who found that speech processing in infancy predicted later reading acquisition in early school age.

Speech perception problems have been thought to be related to inability of processing auditory stimuli. Impaired perception of brief sounds and transitions has an adverse effect to the perception of speech. However, Ramus (2003) has proposed that auditory

deficits in dyslexic individuals are restricted only to a small proportion of dyslexic population and that lower level auditory processing deficits may not be the cause for the higher-level speech perception problems. Tallal (1980) has showed that individuals with language learning disorders show deficits in processing auditory stimuli, especially when stimuli are presented in rapid sequences. Thus, dyslexia might be linked with a weakened ability to discriminate temporal features of stimuli and dyslexia is thought to be due to a general auditory deficit (Rey, De Martino, Espesser, & Habib, 2002; Tallal, 1980). The temporal structure of stimulus can be changed for example by varying the duration of speech or auditory stimuli, and that is one way to study temporal processing of speech.

Because dyslexia is genetically transmitted deficit, the children of dyslexic parents are at an increased risk to develop reading difficulties. Thus, it could be possible to find some differences between children at-risk for dyslexia and normal, healthy children in brain responses already before it is possible to study the reading ability by behavioural tests. In this study, subjects at-risk for dyslexia and control subjects are compared at the age of 6 months and at the age of 6,5 years regarding to their ability to process temporal features of speech. This is made by varying the duration of the consonant /t/ in *ata* pseudowords and by measuring the brain responses with event-related potentials (ERPs) (Leppänen et al., 2002).

1.2 Auditory event-related potentials (ERPs) and their development

Auditory event-related potentials are widely used in the studying of developmental language-related problems (for a review, see Leppänen & Lyytinen, 1997). Event-related potentials are brain responses that are temporally related to an event presented to a subject (Coles, Gratton, & Fabiani, 1990). ERPs can be extracted from electroencephalogram (EEG) data by averaging the time epochs that are related to a certain stimulus type. ERP waveform contains both positive and negative peaks that are named according to their polarity and latency. In addition, these components are differently distributed on the scalp.

The components occurring 50 ms after the event are called long-latency potentials. These potentials are classified into exogenic and endogenic components (Coles et al., 1990; Gevins & Cutillo, 1986.) Exogenic components are also called sensory or obligatory responses and they occur to physical features of the stimuli. Endogenic components, on the contrary, are not automatic but need further information processing to occur. According to Näätänen (1992), exogenic components are quite stable but endogenic components vary more by the internal state and the behaviour of an individual. The scalp distribution of the exogenic components depends on in which sensory modality the stimulus is presented (Coles et al., 1990). For example an auditory stimulus causes predominant activation on the temporal areas of the brain, where the auditory areas are located.

By now, event-related potentials have been well studied in adults. The auditory ERP waveform of a normal adult contains usually exogenic peaks P1 (latency 50 ms), N1 (100 ms), P2 (180 – 200 ms) and an endogenic peak N2 (after 200 ms) (Coles et al., 1990; Gevins & Cutillo, 1986; Näätänen, 1992). Ceponiene, Rinne, & Näätänen (2002) have proposed, that the N1 peak is linked with the auditory sensitivity and orienting, whereas the first positive obligatory deflection P1 and the second negative deflection N2 reflect the auditory sensory processes. N1 deflection has been supposed to consist of three subcomponents: component generated in the auditory cortex on the supratemporal plane (fronto-central), component generated in the association cortex on the lateral aspect of the temporal and parietal cortex (T-complex), and component generated in the motor and premotor cortex (Näätänen & Picton, 1987). N1 may represent an onset response, because it occurs to the onset of the stimulus. It is quite sensitive to the stimulus rate, so that its amplitude starts to decrease after ISI is extended over 10 s. N1 is largest for the first stimulus in a stimulus train (Näätänen, 1992). Attention isn't necessary for the elicitation of N1, but it may increase the amplitude (Näätänen & Picton, 1987).

Although ERPs are well studied in adults, the maturation of the auditory ERPs from infancy to childhood is complex and it needs more research. According to Courchesne (1990), the latencies and amplitudes of ERP peaks do not mature gradually. In addition, ERP components have different developmental time courses and there isn't a hierarchy

in the development of the peaks. In other words, exogenic components do not develop before endogenic components (Courchesne, 1990). Exogenic components change with time so that the complexity of the wave and the amount of the components increase (see for a review Cheour, Leppänen, & Kraus, 2000). In addition, latencies tend to decrease and the polarity of a response may change in prenatal term. The ongoing myelinization is not the only cause to the reduction of ERP latencies in infancy and in childhood. In addition, the synaptic loss is not a cause for the amplitude decrease. The changes in ERP component-structure are due to the complex processes of sculpturing the efficient neural networks for higher stimulus-specificity, automaticity of stimulus processing, maturation of adaptive and attention-related processes, emerging formation of associations and learning and memory (Courchesne, 1990).

In the study of Kushnerenko et al. (2002a) the auditory ERPs were studied from the birth to the age of 12 months using an ISI of 700 ms and three-partial harmonic tones. A positivity called P150 existed already at the age of 3 months, and there wasn't a significant growth of the amplitude from 3 months to 12 months. On the contrary, the latency of P150 decreased from 3 months to 9 months. According to them, the P150 component was a precursor of the mature adult component P1. After the P150 there existed a negativity called N250 and it was thought to represent the adult response N2. The latencies of N250 stayed stable, but the amplitudes increased as a function of age. There wasn't a sign of the precursor of the adult N1 response during the first year of life in their study.

Neuromagnetic responses to tones and pseudowords were measured in adults and children aged 3 months to 15 years in the study of Paetau, Ahonen, Salonen, & Sams (1995). Three ISIs (900 ms, 1200 ms and 2400 ms) were used. Responses were similar to both stimulus types. In the 3-month-old infant P1m (magnetic counterpart of P1) peaked at 190 ms and dominated the response up to 500 ms. In children up to 12 years the P1m was a large deflection starting at 100 ms. After the P1m there occurred a negativity at about 260 ms, which was called N1,2m. When ISI was extended to 1200 ms and 2400 ms an adult type N1m deflection appeared in the most children. It was observed even in a 5-year-old child with the latency of 200 ms. Latencies of all

components tended to decrease with age and the decrease was most rapid before the school age.

Ceponiene, Rinne, & Näätänen (2002) studied children aged 4 and 9 years old and young adults using short stimulus onset asynchrony (SOA) of 700 ms and long SOA, mean of which was 5 s. Stimuli were 100 ms long harmonic tones. In the short SOA condition the P1 predominated around the vertex in both children groups. From the age of 4 to the age of 9 years the amplitudes of the P1 component increased. P1 amplitudes in the adults' group were smaller than in either child group. There wasn't a significant difference in the P1 latency between 4 and 9 years. At both ages the latencies were around 110 ms. In the short-SOA condition there wasn't an N1 deflection in either of the children groups. In the long-SOA condition, which was presented only to the 9-year-olds and adults, there existed the N1 wave in children with the latency of 155 ms. Thus, lengthening the SOA caused the occurring of the N1 in the group of 9 years old. The N2 was robust in both children groups and its amplitude decreased with age. Latencies were around 290 ms at both ages and the N2 was frontocentrally distributed. Ceponiene et al. (2002) suggested that the P1 and N2 reflect the auditory sensory processes and the N1 could be related to the auditory sensitivity and orienting.

Central auditory system maturation was evaluated by Ponton, Eggermont, Kwong, & Don (2000) using multi-electrode recordings of ERPs. Subjects were between 5 and 20 years of age. At the age range 5 – 7 years the P1 was very broad with the latency of about 80 ms. Both the latency and the amplitude of the P1 decreased as a function of age. A component called N1b became detectable first at the age of 9 and it was only a small inflection of the P1 peak. The latency of the N1b was about 100 ms. The N2 occurred in 5 to 7-years-old children with the latency of 200 ms.

Rojas, Walker, Sheeder, Teale, & Reite (1998) studied 6 to 8 years old children's M100 responses (the magnetic analog of the N100) to tone-bursts using ISIs that changed from 2 to 12 seconds. The amplitude of the M100 increased linearly as the ISI was lengthened. The results showed that the M100 refractory period is longer in young children than in adults i.e. the ISI should be long enough for the M100 to appear in

young children. These results were consistent with the results of Paetau et al. (1995) and Ceponiene et al. (2002) mentioned above.

Sharma, Kraus, McGee, & Nicol (1997) studied children ranging in age from 6 to 15 years and adults using synthesized consonant-vowel syllables. There existed a large P1 response at about 100 ms and a later negativity (termed N1b) at about 200 ms in children. In the age range of 6 to 6.9 years the mean latency of the P1 was 87 ms and the N1b was 221 ms. Besides, an earlier negativity called N1a (135 ms) existed in 61% of 6-7 years old, the frequency of occurrence increasing with age. The researchers concluded that the P1 develops systematically into the adult response, but the link of N1a and N1b to the adult N1 was unclear.

By using tone stimuli with three different ISIs (350, 700 and 1400 ms) Ceponiene, Cheour, & Näätänen (1998) studied children aged 7 to 9 years old. The P100 was clear in all ISI-conditions. From the middle ISI to the longest ISI the amplitude decrease was statistically significant. The latency of the P100 (about 100 ms) was not affected with the ISI length. An N160 deflection N160 (with the latency of 160 ms) occurred with the two longest ISIs and the authors suggested it to be a correlate of the adult auditory N1, because a high susceptibility to the stimulus presentation rate, the long refractory period and the frontocentrally dominant scalp distribution indicated a strong link between the N160 and the adult auditory N1 wave. The amplitude and the latency of the N250 (with the latency of 250 ms) didn't change significantly with age. This deflection was suggested to correspond with the N2 in adults.

In the study of Tonnquist-Uhlen, Borg, & Spens (1995) children aged 8 to 16 years were presented tone bursts with the ISI of 1 s. All subjects had the responses P1, N1 and N2. The N1 peak latency was approximately 100 ms and it decreased significantly with age.

In summary, the P1 appears as a robust peak in the ERP waveform very early in infancy with the latency of about 150 – 190 ms (Kushnerenko et al., 2002a; Paetau et al., 1995). It dominates the waveform still in childhood and the latency shortens with age. Amplitudes of this first positive response have showed to be stable from the birth to 12

months (Kushnerenko et al., 2002a) and larger at the age of 9 years than at the age of 4 years (Ceponiene et al., 2002). On the other hand, Ponton et al. (2000) reported amplitudes to decrease from the age of 5 years onward. Same way as the P1, the N2 occurs already in infancy and the latency has been reported to be around 250 ms (Kushnerenko et al., 2002a). The latency shortens with age being 200 – 290 ms in childhood (Ceponiene et al., 1998; Ceponiene et al., 2002; Ponton et al., 2000). The N2 amplitude has been reported to increase from the birth to the age of 12 months and decrease from the age of 4 years to the age of 9 years (Ceponiene et al., 2002). Contrary to the P1 and the N2, the deflection N1 has been discovered to not arise until preschool age. When the ISI has been long enough, even the children aged 5 years have showed a sign of the N1 peak (Paetau et al., 1995). Tonnquist-Uhlen et al. (1995) reported all children aged 8 years to show the N1 when the ISI was 1 s. In childhood the latencies of the N1 vary from 100 (Ponton et al., 2000) to 200 ms (Paetau et al., 1995).

1.3 Mismatch negativity (MMN) and its development

The ERP-component mismatch negativity (MMN) is a useful tool in investigating the detection of a change in a stimulus chain. Thus, the temporal processing of speech can be measured with the MMN component. The MMN was first found and described by Näätänen, Gaillard, & Mäntysalo (1978) in a dichotic listening situation. It is a negative wave usually starting 100 ms and ending 250 ms after the stimulus onset in adults, and it is normally frontocentrally distributed on the scalp. In the magnetoencephalography (MEG) study Hari, Rif, Tiihonen, & Sams (1992) found a mismatch field to frequency changes of single tones and paired tones, and the source was located on the supratemporal auditory cortex about 1 cm anterior to the N100m.

The kind of paradigm where deviant stimuli is presented in among the standard stimuli is called an oddball paradigm (Gevins et al., 1986). The elicitation of the MMN to the deviant stimuli can be studied with this paradigm. Usually subjects perform simultaneously a task that fixes their attention away from the presented stimuli. Because this component doesn't need attention to occur, it facilitates the researching infants and

young children compared to the behavioural research, like the sucking paradigm (Näätänen, 1992).

MMN is elicited by any discriminable change in a repetitive stimulus chain. (Näätänen et al., 1978; Näätänen, 1992). So, a rarely occurring stimulus in the chain of frequently presented identical stimuli can elicit the MMN. It doesn't occur to the firstly presented stimulus but needs deviation between stimuli to appear. For auditory stimuli the MMN is related to the change in frequency, intensity, spatial location of a stimulus, risetime, duration, phonetic structure and in partial omission of a compound stimulus (Näätänen, 1992). The cause of the MMN is thought to be either in an activation of new afferent elements in the brain to a deviant stimulus or the mismatch of a deviant incoming stimulus to a memory trace of a repetitive, standard stimulus (Näätänen, 1992). According to the later explanation, the MMN elicitation is based on the presence of the short-term memory trace, which is formed in the auditory cortex while identical stimuli are presented to the subject (Näätänen, 2001). The memory trace has reported to fade within 5-10 s (Näätänen, Paavilainen, Alho, Reinikainen, & Sams, 1987 in Näätänen, 2001). So, the interstimulus interval (ISI) needs to be short enough for the MMN to occur, because when the ISI is too long the MMN first weakens and then disappears (Näätänen, 1992; Näätänen, 2001). Cowan, Winkler, Teder, & Näätänen (1993) have suggested that there are at least two conditions for an MMN elicitation to change in a tone frequency. Firstly, the representation of the standard stimuli must be well established as a standard in memory. Secondly, the representation of the standard stimuli must be in a currently active state.

According to the reviews of Alho & Cheour (1997), Cheour, Korpilahti, Martynova, & Lang (2001) and Cheour, Leppänen, & Kraus (2000) even infants show clear MMN to simple tones as well as phonemes. Besides, MMN has been succeeded to record from preterm infants (Cheour-Luhtanen et al., 1996), which shows it to be the ontogenetically earliest discriminative response of the human brain. The MMN has been found to be developmentally quite stable, but in childhood the latency decreases and the amplitude increases as a function of age. However, children have a larger MMN amplitude than adults. The scalp distribution of the MMN differs between adults and children. Instead of frontocentral distribution in adults, a prominent MMN can be obtained in infants also

at parietal scalp sites. Uwer & Suchodoletz (2000) have suggested that the MMN might not be useful as an individual diagnostic measure in children, because in their study the MMN showed a decrease of amplitude during the second test session in the late latency window (400 – 500 ms) for the frequency and speech elicited MMN. On the other hand, the duration deviant showed individual stability.

In the study of Cheour et al. (1998) MMN was compared in pre-term and full-term newborns and in infants aged 3 months by using Finnish vowels /y/ and /i/. There was no significant difference in the MMN amplitudes between the groups, but the mean MMN latency decreased significantly as age increased. In the age of 3 months, MMN started to peak at the latency of 150 ms and its mean peak latency was 229 ms.

Kushnerenko, Ceponiene, Balan, Fellman, & Näätänen (2002b) studied the development of the pitch change detection in infants from birth to 12 months every 3 months. The MMN existed in all age groups with the latency of 150 – 250 ms. MMN was not identifiable in some infants from age to age and the authors explained this by some other factors, like MMN, overlapping with the subsequent positivity especially at the ages of 3 and 6 months.

Infants between 2 and 6 months of age were studied by Trainor et al. (2003). They found that infants at the age of 2 months had only a positive slow wave to the deviant stimulus that differed from the standard by containing a short silent gap. By the age of 6 months most of the infants showed negativity at approximately 200 ms and it closely resembled the MMN of adults.

The change detection of different word stress patterns was studied by Weber, Hahne, Friedrich, & Friederici (2004). Different stimuli were formed by varying the duration of either the first or the second /ba/ of the item /baba/. Subjects were infants aged 4 or 5 months or adults. Adults displayed a typical MMN to the stress on the first syllable as well as to the stress on the second syllable. Infants aged 4 months didn't show reliable discrimination response to different stress patterns, but infants aged 5 months were able to distinguish the stress on the first syllable from the stress on the second syllable. This mismatch response was a positivity at 460 – 540 ms after a negativity, which wasn't

statistically significant. This positive MMR is consistent with the findings of Trainor et al. (2003). Results showed that the processing of different stress patterns relevant for word recognition develops between the age of 4 and 5 months.

MMN was studied in infants aged 8 months and in adults using speech consonants /da/ and /ta/ by Pang et al. (1998). The MMN was present in both age groups but there were differences in the scalp distributions. A clear infant MMN with an onset of approximately 200 ms and duration of 100 – 150 ms was observable only at C3 and T3 electrodes, whereas in adults the MMN was present at Fz, Cz, C3, C4, and Pz. The researchers explained this difference between the age groups by the developmental changes in the neural systems utilized in language processing.

Korpilahti, Krause, Holopainen, & Lang (2001) recorded ERPs to complex sounds, natural words and pseudowords in children aged 4 to 7 years. The stimuli elicited MMN that had two peaks, of which the former (150 – 200 ms) was larger for the sounds and the latter (400 – 450 ms) for the words. The MMN was significantly larger for the words than for the pseudowords. The former MMN was thought to represent acoustical differences and the latter peak cognitive evaluation of the stimuli. Also Dehaene-Lambertz & Baillet (1998) have proposed these differences between the early and the late MMN.

As in the aforementioned study of Trainor et al. (2003) and Weber et al. (2004) in infants, Maurer, Bucher, Brem, & Brandeis (2003a) found a frontal positive mismatch response with posterior negativity in children aged 6 to 7 years. The latency range of this mismatch response was 179 – 207 ms. Adults responded with frontocentral negativity (129 – 199 ms). Stimuli deviated either in the frequency (1000, 1030 and 1060 Hz) or in the phoneme (/ba/, /da/ and /ta/). Deviations were small and the ISI was short (0.38 s) and this was suggested to be the cause for this positive response in children.

Kraus, McGee, Sharma, Carrel, & Nicol (1992) presented phonemes /da/ and /ga/ to children aged 7 to 11 years. MMN appeared in all children and the mean latency of the peak was 230 ms. In their later study, Kraus et al. (1993) investigated MMN once again

in 7 to 11 years old children. They used variants of the voiced stop consonant /da/. MMN was apparent in all the children with the latency of around 200 ms, although the stimuli were just perceptibly different. This showed that the MMN is an extremely sensitive response to physical stimulus differences.

1.4 MMN and language learning deficits

MMN is a useful tool for assessing normal and deficient development of auditory discrimination, sensory memory and speech perception. Studies concerning different language learning deficits have dealt with dyslexia (for a review, see Kujala & Näätänen, 2001), specific language impairment (SLI), dysphasia etc.

Studies concerning developmental dysphasia have showed attenuated MMN to frequency differences (Holopainen, Korpilahti, Juottonen, Lang, & Sillanpää, 1997; Korpilahti & Lang, 1994) as well as to the large duration differences (50 ms vs. 500 ms, in the study of Korpilahti & Lang, 1994) in the dysphasic children compared to the control children. On the contrary, in the study of Uwer, Albrecht, & Suchodoletz (2002) with SLI and control children, children with SLI had problems in automatic processing of different speech stimuli, whereas processing of tone differences, like frequency and duration, was intact. School-aged children with learning problems and normal children were compared by using MMN and behavioural tests as measures in the study of Kraus et al. (1996). They evaluated the discrimination ability of rapid speech changes (/da/ versus /ga/ or /ba/ versus /wa/). Children with learning problems were poorer in behavioural discrimination and there were differences between groups also in electrophysiological responses. The impaired behavioural discrimination and the diminished magnitude of the MMN had a positive correlation. Authors suggested that inability to discriminate rapid speech changes depends not on attention and conscious perception and that there may be a deficit in the auditory pathway.

Dyslexic and control adults were compared using MEG in the study of Helenius, Salmelin, Richardson, Leinonen, & Lyytinen (2002). The spatiotemporal pattern of the auditory cortical activation was studied using natural bisyllabic pseudowords (/ata/,

/atta/ and /a a/), complex non-speech sound pairs (corresponding to /atta/ and /a a/) and simple 1-kHz tones. The results showed that 100 ms after the onset of the vowel /a/ there was a difference between dyslexic and control adults in the supratemporal auditory cortex. This N100m response was abnormally strong in dyslexic individuals. There weren't group differences in amplitudes in the complex non-speech sound and tone conditions. The authors interpreted that there is a difference between dyslexic and normal people as early as 100 ms after the speech onset, and that the N100m response codes stimulus specific features, which are likely to be important for the speech perception.

In the study of Schulte-Körne, Deimel, Bartling, & Remschmidt (1999) MMN was assessed in 15 dyslexic and 20 control adults. Subjects were presented a complex tonal pattern, in which a deviation between standard and deviant stimulus was in the temporal structure. The MMN was significantly smaller in dyslexics in the time window 250 – 600 ms compared to controls. This reinforced the view that it might be the ability of processing the temporal information that is weakened in dyslectic individuals. On the other hand, in the latter study of Schulte-Körne, Deimel, Bartling, & Remschmidt (2001), the attenuated MMN in dyslexic subjects to the speech but not to the tone stimuli was thought to be due to speech perception deficit in dyslectic people.

Kujala, Belitz, Tervaniemi, & Näätänen (2003) studied the auditory change detection in dyslexic and control adults. Dyslexic subjects had an attenuated MMN response compared to controls, when an additional tone followed tone pairs. Response to pitch change was also diminished at the left hemisphere of the dyslexics. The authors suggested that dyslexic individuals have problems in temporal discrimination and lowered tolerance for backward-masking effects. These auditory processing problems were thought to be due to a deficit at a very early information processing stage in the cortex or even prior to that in the subcortical structures.

Leppänen, Pihko, Eklund, & Lyytinen (1999) compared infants with increased risk for dyslexia and control infants after birth and at the age of 6 months. Standard and deviant syllable stimuli differed in the length of the vowel /a/ (/kaa/ was standard and /ka/ was deviant). Groups differed as early as in newborns in their ability to process duration of

speech. Leppänen et al. (2002) studied at-risk and control infants at the age of 6 months. The ERPs generated by the changes in the temporal structure of the pseudowords /ata/ and /atta/ were measured. The pseudoword /atta/ differed from /ata/ so that the duration of the consonant /t/ was longer in /atta/. The at-risk infants responded more sensitive to sounds per se than the control infants i.e. they had larger obligatory positive responses than controls. In addition, the at-risk infants showed a diminished MMN response to duration changes in speech. This indicated a deficit in temporal processing of speech in infants at-risk for dyslexia.

Children in at-risk for dyslexia and control children aged 6 to 7 years were compared in the study of Maurer, Bucher, Brem, & Brandeis (2003b). Stimuli were the same frequency and phoneme deviants as in the above-mentioned study of Maurer et al. (2003a). The stimuli were 100 ms in duration and were presented with the SOA of 383 ms. The at-risk children responded more positively at the latency range of 109 – 140 ms especially to phonemes, and the late MMN response at the latency range of 457 – 636 ms was attenuated in at-risk children especially for tones. The authors suggested that these results supported the basic auditory processing deficit in children at risk for dyslexia.

In summary, differences in MMN responses between dyslexic and control subjects have been found both in the lower level auditory processing and in the higher level phonological processing. As above-mentioned studies by Leppänen et al. (1999 and 2002), this study also belongs to the Jyväskylä Longitudinal Study of Dyslexia (JLD). The first purpose of this study is to research the development of ERP deflections in infants and preschool children from the age of 6 months to the age of 6,5 years. Especially the amplitudes and the latencies of positive and negative obligatory deflections will be examined. Leppänen et al. (2002) found differences between at-risk and control infants in temporal auditory processing of speech changes. Thus the second question in this study is, if the differences between groups in automatic discrimination ability of temporal changes in pseudowords /ata/ and /atta/ will remain until 6,5 years and if there will be new differences between groups in childhood.

2. METHODS

2.1 Participants

52 healthy children without hearing deficit or neurological disorder participated in this study. Same individuals were first studied in infancy, when the mean age was 6 months and 7 days and then in childhood, when the mean age was 6 years, 6 months and 10 days. All the subjects were from families, which were recruited, according to institutional informed consent procedures for the Jyväskylä Longitudinal Study of Dyslexia. Of the subjects, 26 (13 boys) were from families with a familial background of dyslexia and belonged to *the at-risk group*. 26 of the subjects (17 boys) were from matched control families without any signs of dyslexia, and they belonged to *the control group* (for details on the participant characteristics and selection criteria, see Leinonen et al., 2001). The parents in both groups reported no hearing problems, nor any sensory or neurological abnormalities.

The inclusion criteria for the at-risk group were either parent's report of his or her own reading disorder, a comparable report concerning at least one close relative, and multiple diagnostic test results indicative of dyslexia. To be diagnosed with dyslexia, the parent had to have a score at least 1 SD below the norm in accuracy or speed of oral text reading, or in accuracy of written spelling, and also in at least two separate single-word measures (either accuracy or response latency of word recognition, pseudoword decoding or lexical decision). Their IQ had also to be 85 or above (assessed with the Raven B, C, and D matrices; Raven, Court, & Raven, 1992). Some of the parents who had several relatives with a reading disability, despite self-reported school age and present reading problems, did not score significantly below the norm on all the required diagnostic measures. However, because of their strong family history of reading difficulties, they were included as compensated dyslexics (it should be noted that inclusion of the families with compensated dyslexics was a "conservative" decision, in that their infants/children could only be expected to differ to a lesser degree than other at-risk infants/children from control infants/children). The mothers' educational status,

as determined on the basis of the length or level of education, or their IQ, did not differ between the groups when subjects were infants.

2.2 Stimuli and procedure

Three naturally produced pseudowords were used as stimuli. They comprised the short *ata*, which had a voiceless stop with a silent period of 95 ms in the middle of the sound (with a total duration of 300 ms; the duration of the first part of the stimulus, the initial glottal stop and /a/ vowel together, was 72 ms, and that of the second part, including the explosion of /t/ and the final /a/ vowel, was 133 ms), an intermediate *ata*, and the long *atta*. The latter two stimuli were produced by lengthening silent gap to 195 and 255 ms, respectively. All other acoustical aspects, such as fundamental frequency and intensity, were held constant (for details; see Leppänen et al., 2002; Pihko, Leppäsaari, Leppänen, Richardson, & Lyytinen, 1997; Richardson, 1998; Richardson et al., 2003). ERP data reported here are for the short *ata* and the long *atta*.

MMN-paradigm. Stimuli were presented via a loudspeaker in short sequences with an intensity of 75 dB SPL (sound pressure level) in two different conditions: In the Long atta deviant condition (data from 17 at-risk and 19 control subjects at both ages), the short *ata* was presented as the frequent "standard" stimulus, with an 80% probability of occurring. The intermediate and the long *atta* stimuli were presented as rare deviant stimuli on 10% of trials for each. The stimulus sequence in the Short ata deviant condition (data from 9 at-risk children and 7 control children; in infancy results are reported by Leppänen et al. (2002), n = 12 and 12 for the at-risk and the control infants, respectively) was similar to the first one, except that the long *atta* occurred on 80% of trials (as the standard) and the short *ata* on 10% (as the other deviant). This reversed condition was used to find out whether the stimuli as such or their difference determined ERP and related group differences. The fixed offset-to-onset interstimulus interval (ISI) was 610 ms in both conditions. In infancy also ISI of 450 ms was used in the Short ata deviant condition, but as no statistical differences between two ISIs were found in the reported ERP measure in the control group, the data were pooled across the two ISI conditions (Leppänen et al., 2002). In this study, for the Short ata deviant

condition only children aged 6,5 years were studied. For the results of 6-month-old infants Leppänen et al. (2002) will be reviewed.

2.3 EEG recording and ERP averaging

<u>During the EEG recordings</u> infants were seated on their parent's lap and children sat on a chair in an electroencephalogram (EEG) laboratory room. EEG/ERPs were recorded from 13 scalp sites, F3, F4, Fz, T3, T4, C3, C4, Cz, P3, P4, Pz, O3, O4 according to the international 10–20 electrode system with Ag/AgCl electrodes attached in an EEG-cap (Blue sensor, Medicotest, Denmark) and referred to the ipsilateral mastoid electrodes (for a part of the participants, bipolar derivations between the left and the right hemispheres, F3-F4, C3-C4, and P3-P4, were used instead of the midline z-channels, but neither the bipolar derivations nor these z-channels were included in the analyses in any subject). Electro-oculograms (EOGs), referenced to the left mastoid, were recorded, with one electrode lateral to and slightly above the left eye, and another lateral to and below the right eye. The online filtered (passband 0.5 – 35 Hz, sampling rate 200 Hz) EEG epochs of -50 – 840 ms (with a 50 ms prestimulus baseline) were averaged offline separately for each stimulus type. Epochs with artefacts, deflections exceeding ± 200 µV (for infants) or ± 150 µV (for children) at EOG (excessive eye movements) and EEG channels (muscle activity or other extra cerebral artefacts) were excluded from the averaging. The mean number of acceptable EEG epochs for the deviant stimulus at the age of 6 months in the Long atta deviant condition was 106 (range = 79–137) in the at-risk group and 103 (range = 73–133) in the control group. At the age of 6,5 years the acceptable epochs for the deviant stimulus were 99 (range = 70–113) and 94 (range = 69–124), respectively. In the Short ata deviant condition the number of accepted EEG epochs for deviant stimulus were 99 (range = 77–116) in the at-risk group and 96 (range = 72–121) in the control group at the age of 6,5 years. Subjects with less than 65 acceptable EEG epochs were excluded from this report. The number of accepted EEG epochs for standard stimuli were approximately three times the number of deviant stimuli, because only three last standard epochs before the deviants were accepted for averaging.

2.4 Data analysis

The ERP measures at 6 months and at 6,5 years. The mean amplitudes around and the latencies of the major peaks were used as the ERP measures. Time windows for analysing major peaks were selected on grounds of grand average waveform. The measures for the 6-month-olds were the same as used in the study of Leppänen et al. (2002). The selection of the time windows for 6,5 year olds was done by the present author and Leppänen. To make sure that individual peaks fitted into the selected time periods, the present author checked individual waveforms. In some cases individual adjustments in time windows were made for including the peaks that were too early or late compared to grand average waveform. This made the statistical comparisons reasonable. Individual mean amplitude values were calculated over the 30-ms period centered at each major peak in the waveform. Individual peak latency values were also calculated. The correctness of data was checked firstly by making sure that the ERP-waveform and the calculated amplitude and latency values matched, and secondly that these values and statistical SPSS-data matched.

At the age of 6 month in the Long atta deviant condition the peaks for standard stimuli were N80, P190, N320, N450, P470 and P840 (deflections named according to polarity and approximate peak latencies). For deviant there was an additional peak N600. At the age of 6,5 years the peaks for standard stimuli were N40, P110, N225, P250, N310 and N450 (this was N600 for deviant). Peaks that were selected for statistical analyses were $P_{i190/c110}$, $N_{i320/c225}$, and $N_{i450/c450}$ (capital letter means polarity, subindex letter i is for infancy and c is for childhood, number means the latency for standard stimuli). In the Short ata deviant condition the deflection N_{c450} was analysed. O-channels (O3 and O4) were left outside analyses, because the responses in these channels were weak and the auditory activation is not thought to extend the occipital regions of the scalp.

Statistical analyses. Development of ERP deflections and possible developmental changes in the ERP measures between at-risk and control subjects from the age of six months to the age of six and half years were statistically analysed using Age (6 months vs. 6,5 years) × Stimulus type (standard vs. deviant) × Scalp site (8 electrodes) × Group

(risk vs. control) multiple analyses of variance (MANOVAs) for repeated measures for each deflection separately. Responses at different hemispheres and at anterior (frontal and central electrodes) versus posterior scalp sites were studied using Age × Hemisphere (left vs. right) × Anterior-posterior Scalp site (F, C and P electrodes) × Group MANOVAs for repeated measures. These were made for each deflection and for standard and deviant stimuli separately. Above mentioned analyses were also made for peak latencies. When appropriate, results from the repeated measures analyses of variance (ANOVAs), being sometimes more sensitive than MANOVA, are reported with the corrections for sphericity if applicable.

Some analyses were made for infants and children separately, because in this study the number of subjects was smaller than in the study of Leppänen et al. (2002). The purpose was to make sure which group differences were remained from infancy to childhood and if there were new differences between groups in childhood compared to infancy. Only amplitudes were analysed for these group differences. A Stimulus type (standard vs. deviant) × Scalp site (8 electrodes) × Group (risk vs. control) multiple analyses of variance (MANOVAs) for repeated measures for each deflection separately were made. Besides, all peaks were analysed separately for standard and deviant stimuli using Scalp site (8 electrodes) × Group repeated measures MANOVA. In addition, these analyses were made for both the hemispheres separately.

For comparing the responses to the short *ata* in the Long atta deviant condition and in the Short ata deviant condition a Scalp site (8 electrodes) × Stimulus type (*ata* as standard vs. *ata* as deviant) repeated measures MANOVA were made for each group separately. The responses between groups were also compared using Mann-Whitney U nonparametric test for two independent samples. This test was used because the groups in the Short ata deviant condition were too small, (n = 7 and 9 in the control and in the at-risk groups, respectively) for the parametric statistics. These analyses were made for each electrode site separately.

3. RESULTS

3.1 Waveforms in the Long atta deviant condition

Leppänen et al. (1997) have already reported how the waveform was in the group of 6 months old infants. Now the group of subjects was smaller (n = 36, 19 in the control group and 17 in the at-risk group) than in the study of Leppänen et al. (n = 52), but the waveform was similar in both situations. At the age of 6 months (Figures 1a and 1b) a small negative deflection N80 was followed by a widespread positive-negative-positive waveform pattern (P190, N320, P470). The response to the long *atta* was mainly in one way markedly different from that to the short *ata*. For the long *atta*, the major negative deflection at about 310 – 330 ms (N320) was shorter in duration. In addition, for the long *atta* there existed another late negative peak at 600 ms (N600).

Although the waveform was similar in both of the groups, the response to the deviant long *atta* seemed to be throughout more negative in the control group than in the at-risk group, especially at the central and temporal electrodes C3, C4, T3 and T4, and at the parietal electrode P3 at the left hemisphere (Figure 1c).

FIGURE 1. ERPs (averaged across infants) at the age of 6 months in the Long atta deviant condition. **(A)** Responses to standard *ata* vs. deviant long *atta* in the control group (n = 19). **(B)** Responses to standard *ata* vs. deviant long *atta* in the at-risk group (n = 17). **(C)** Responses to deviant long *atta* in the control group vs. at-risk group. Responses to deviant stimuli are more negative in the control group compared to the at-risk group. Stimuli are represented with small boxes on the calibration line. Negativity up.

At the age of 6,5 years (Figure 2a and 2b), there existed same peaks for the short *ata* and the deviant long *atta* in the ERP waveform, but these peaks differed only a bit for different stimulus types. The first small deflection was a negative N40. It was followed by P110, N225, P250 and two negative deflections N310 and N450 (latencies are for the standard stimuli). For the standard stimuli, the N225 was a very weak peak that seemed to be only a part of the slope of the following negative deflection N310. However, for the deviant long *atta* the deflection N225 was markedly larger and lasted longer than for the standard short *ata*. The deflection P250 occurred after that, and it was also larger for the long *atta* than for the short *ata*. The positive P250 and the following negative deflections peaked markedly later for the long *atta* than for the short *ata*, otherwise the waveforms were similar for these two stimulus types. The later occurrence of the above mentioned peaks for the long *atta* was probably caused by the longer silence gap between the first and the second part of the stimulus and the later starting point of the stimulus part /ta/ in the deviant stimulus.

As in infancy, the waveform for the deviant long *atta* was very similar in both of the groups at the age of 6,5 years (Figure 2c). Again, there existed small differences in negativity of the waveform between the groups. The late negativity (N600 for deviant) seemed to be a bit more negative in the control group than in the at-risk group. This difference was best seen at the left hemisphere at the central and parietal electrodes (C3 and P3).
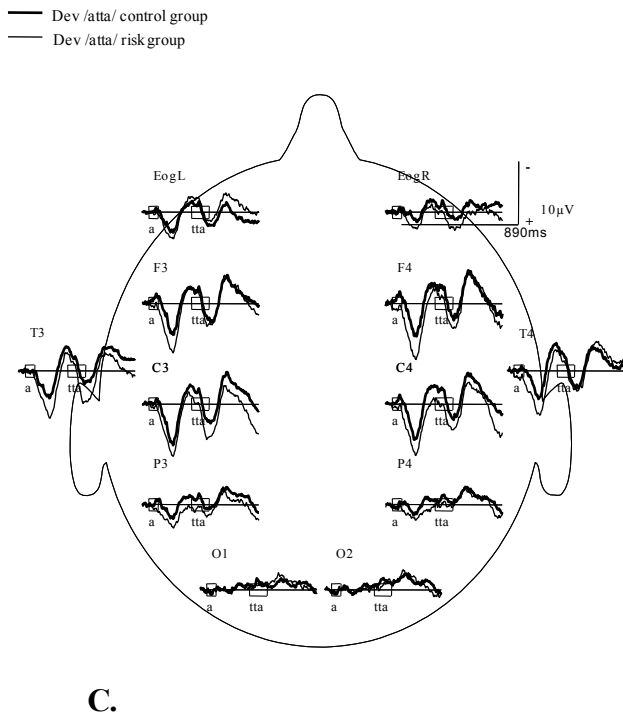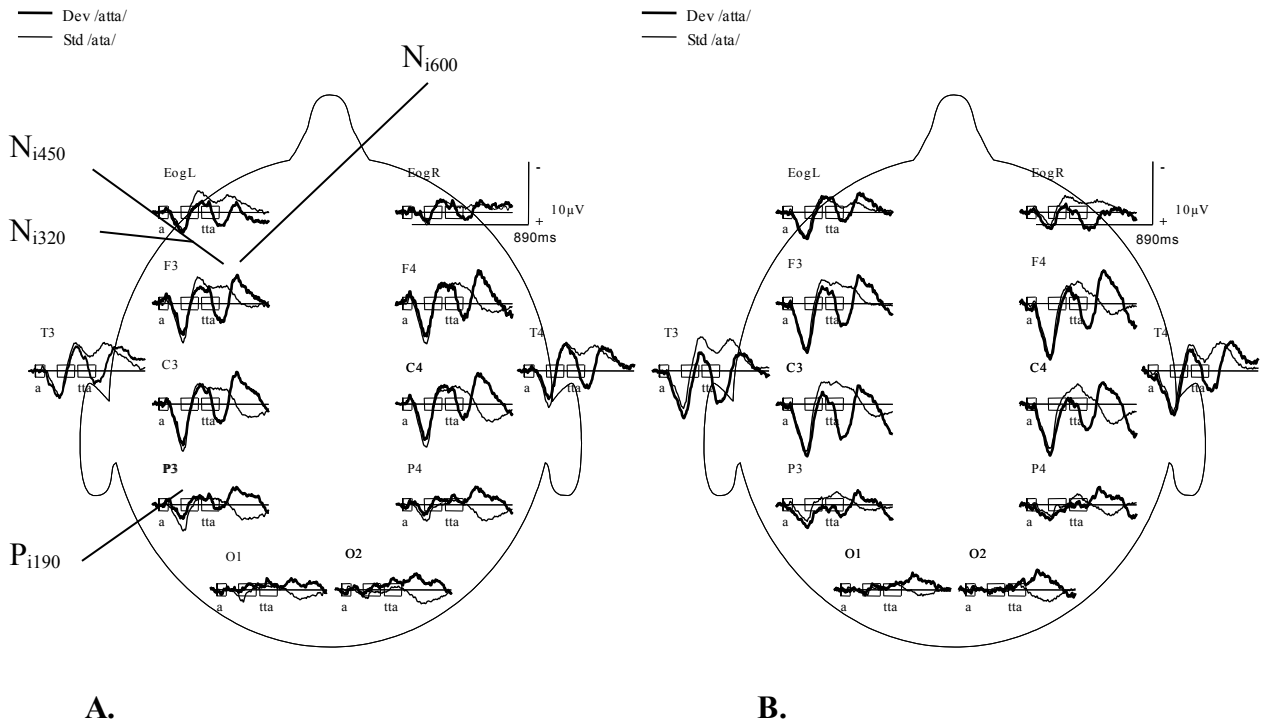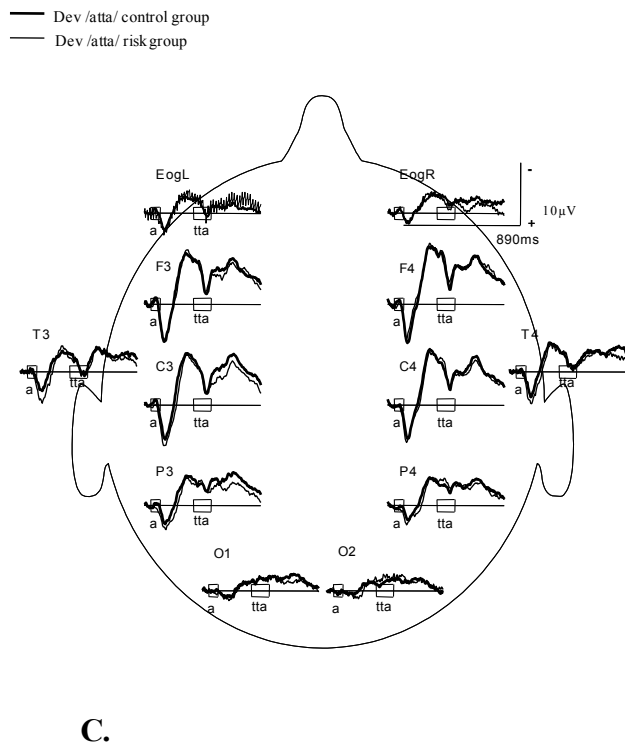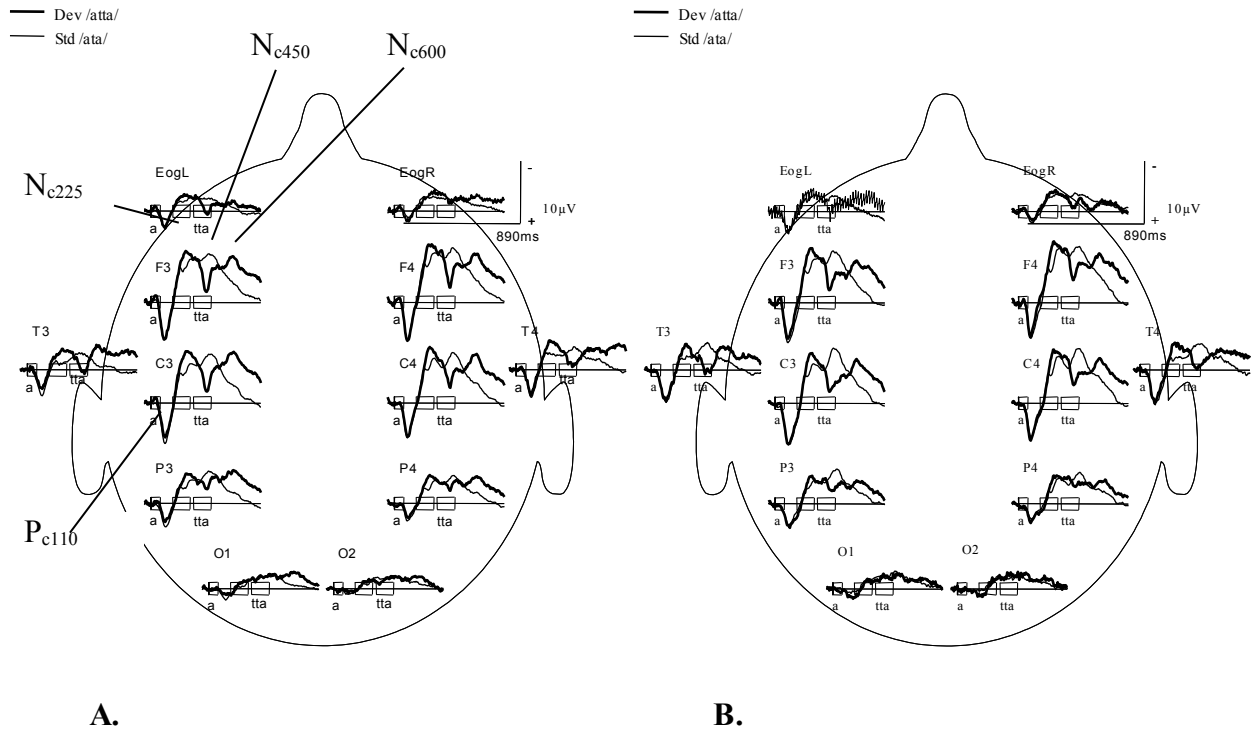
FIGURE 2. ERPs (averaged across children) at the age of 6,5 years in the Long atta deviant condition. **(A)** Responses to standard *ata* vs. deviant long *atta* in the control group (n = 19). **(B)** Responses to standard *ata* vs. deviant long *atta* in the at-risk group (n = 17). **(C)** Responses to deviant long *atta* in the control group vs. at-risk group. Responses to deviant stimuli seem to be a bit more negative at the central and parietal scalp areas at the left hemisphere in the control group compared to the at-risk group. Stimuli are represented with small boxes on the calibration line. Negativity up.

3.2 Positive deflection $P_{i190/c110}$ in the Long atta deviant condition

Developmental changes in amplitudes. Mean amplitude values for the positive obligatory response were analysed by an Age × Stimulus type × Scalp site × Group multiple analysis of variance (MANOVAs) for repeated measures. The main effect of Age was significant, $F(1, 34) = 4.60$, $p < .039$, indicating that response to this positivity was different at different ages. Means (Appendix, Tables 1 and 2) showed that amplitudes were larger in infancy than in childhood. There was also significant interaction Age × Scalp site, $F(7, 28) = 4.26$, $p < .003$, $\Lambda = .485$, showing that the topographical distribution of the scalp was different at different ages. Scalp site changes between ages led to more detailed analyses of Age × Hemisphere × Anterior-posterior scalp site × Group for both stimulus types separately.

Standard and deviant stimuli. For the ERPs to the standard stimuli there was a significant Hemisphere effect, $F(1, 34) = 4.78$, $p < .036$. Means were usually more positive at the left hemisphere across both ages and groups. There were also developmental changes between hemispheres in anterior-posterior responses (Age × Hemisphere × Anterior-posterior), $F(2, 33) = 4.15$, $p < .025$, $\Lambda = .798$. In infancy amplitudes at right frontal electrode were larger than at left frontal electrode but in childhood all amplitudes were larger at the left hemisphere. Age had an effect on the anterior-posterior topographical distribution (Age × Anterior-posterior) across both groups, $F(2, 33) = 11.05$, $p < .000$, $\Lambda = .598$. This was due to the reduction of amplitudes at the anterior scalp sites from infancy to childhood, while the posterior scalp sites showed, in contrast, larger amplitudes in childhood than in infancy. For the ERPs to the deviant stimuli there was a significant interaction Age × Anterior-posterior, $F(2, 33) = 6.96$, $p < .003$, $\Lambda = .703$. This indicated that the anterior-posterior topographical distribution was different at different ages across both hemispheres and groups.

Differences in amplitudes between the control and at-risk groups. An Age × Stimulus type × Scalp site × Group MANOVA gave a Stimulus type × Group interaction, $F(1, 34) = 5.68$, $p < .023$, as well as an Age × Stimulus type × Group interaction, $F(1, 34) =$

4.98, p < .032. This indicated that there were differences between the groups in responses to standard versus deviant stimuli across both ages, and that the interaction between age and stimulus type (standard vs. deviant) differed between the groups. At the age of 6 months, amplitude means were more positive for the standard than for the deviant stimuli in the control group. In the at-risk group differences between stimulus types were contradictory. At the age of 6,5 years also the at-risk group responded usually more positively to the standard than to the deviant stimuli (Pictures 1 a, 1b, 2a and 2b; Appendix, Tables 1 and 2). In the separate analysis for infants, a Stimulus type × Scalp site × Group MANOVA showed a significant Stimulus type × Group interaction, $F(1, 34) = 7.63 < .009$. In the separate analysis for children there wasn't a Stimulus type × Group interaction any more, but there was a Scalp site × Group interaction at the left hemisphere, $F(3, 32) = 4.12, p < .014$.

Standard and deviant stimuli. For the ERPs to the standard stimuli, groups differed in their anterior-posterior scalp topography (Anterior-posterior × Group), $F(2, 33) = 3.73$, $p < .035, \Lambda = .815$. The at-risk group responded more positively at the frontal than at the central channels, whereas responses in the control group were contradictory. There were also developmental changes in the anterior-posterior scalp topography between the groups (Age × Anterior-posterior × Group), $F(2, 33) = 3.52, p < .041, \Lambda = .823$. The separate analysis for infants (a Scalp site × Group MANOVA) showed a Group main effect at the right hemisphere, $F(1, 34) = 5.55, p < .024$, which indicated that groups differed in their responses to the standard stimuli at the right hemisphere across all the scalp sites in infancy. There was also a significant interaction Scalp site × Group, $F(7, 28) = 2.64, p < .031, \Lambda = .602$. In the separate analysis for children there wasn't any significant differences between the groups for standard stimuli. For the ERPs to the deviant stimuli there was an Age × Group interaction, $F(1, 34) = 4.43, p < .043$, indicating that there were developmental changes between the groups in responding to the deviant stimuli. The amplitude means showed that the responses of the at-risk infants were more positive than the responses of the control infants. These differences between the groups disappeared in childhood, (Figures 1c and 2c, Appendix Table 2). A significant ANOVA effect Age × Anterior-posterior × Group, $F(2, 68) = 4.38, p < .016$, indicated that the effect of age was different between groups for the anterior-posterior

scalp topography. It seemed that this was caused by decreasing amplitudes at all the six electrodes in the at-risk group from the age of 6 months to the age of 6,5 years, whereas in the control group responses at the frontal and parietal electrodes were larger in childhood than in infancy. The separate analysis for infants (a Scalp site × Group MANOVA) found a significant Group main effect, $F(1, 34) = 7.59$, $p < .009$, which indicated that the groups differed across all the scalp sites in their responses to the deviant stimuli in infancy. In the separate analysis for children there was a significant interaction Scalp site × Group at the left hemisphere, $F(3, 32) = 3.27$, $p < .034$, $\Lambda = .765$. In the control group the responses were largest at the frontal and smallest at the temporal electrodes. In the at-risk group the responses were largest at the central and smallest at the parietal electrodes.

Developmental changes and differences between the control and at-risk groups in latencies. Peak latencies were analysed in the same way as above. An Age × Stimulus type × Scalp site × Group MANOVA gave a significant Age effect, $F(1, 34) = 581.33$, $p < .000$. Latencies shortened from infancy to childhood (Appendix Tables 3 and 4). A significant Stimulus type × Scalp site × Group interaction, $F(7, 28) = 3.27$, $p < .011$, $\Lambda = .549$, indicated that the speed of the response to the standard versus deviant stimuli changed differently between the groups at different scalp sites across both age-groups.

3.3 Negative deflection $N_{i320/c225}$ in the Long atta deviant condition

Developmental changes in amplitudes. For the negative deflection $N_{i320/c225}$ an Age × Stimulus type × Scalp site × Group repeated measures MANOVA gave a significant Age main effect, $F(1, 34) = 12.53$, $p < .001$, and this was due to more negative amplitudes in childhood than in infancy (Pictures 1a, 1b, 2a and 2b; Appendix, Tables 5 and 6). There was a Stimulus type main effect, $F(1, 34) = 25.38$, $p < .000$, which indicated that the responses to the standard and deviant stimuli deviated significantly from each other across both ages and all scalp sites. A significant Age × Stimulus type interaction was also found, $F(1, 34) = 78.19$, $p < .000$. These effects were a result of more negative responses to the standard than to the deviant stimuli in infancy and

opposite responding between stimulus types in childhood (separate analyses for infants and children, a Stimulus type × Scalp site × Group MANOVA, showed a Stimulus type main effect in infancy at the left hemisphere, $F(1, 34) = 4.68 < .038$, and at both hemispheres in childhood $F(1, 34) = 91.84$, $p < .000$). A significant Age × Scalp site interaction was found, $F(7, 28) = 25.37$, $p < .000$, $\Lambda = .136$. This indicated that topographical distribution changed with age. A significant Greenhouse-Geisser corrected ANOVA effect for Stimulus type × Scalp site, $F(7, 238) = 3.31$, $p < .011$, indicated that different stimulus types were responded differently at different scalp sites across both ages and groups. In addition, there were developmental changes in aforementioned responses (Age × Stimulus type × Scalp site), $F(7, 28) = 3.79$, $p < .005$, $\Lambda = .514$. Age driven differences in response amplitudes at different scalp locations as well as between those to deviant versus standard stimuli led again to further studies using Age × Hemisphere × Anterior-posterior scalp site × Group analyses for ERPs to standard and deviant stimuli separately.

Standard and deviant stimuli. For the ERPs to the standard stimuli there was a significant interaction Age × Hemisphere, $F(1, 34) = 4.42$, $p < .043$. Amplitude means showed that at the age of 6 months responses were more negative at left than at the right hemisphere but in childhood the hemisphere differences were contradictory. Anterior-posterior scalp topography differed between hemispheres at different ages (Age × Hemisphere × Anterior-posterior), $F(2, 33) = 7.03$, $p < .003$, $\Lambda = .701$. This was due to more negative amplitudes at anterior electrodes at the left hemisphere than at the right hemisphere in infancy, whereas in childhood these response differences between hemispheres were reversed. Responses stayed more negative at left than at the right hemisphere only at parietal scalp sites from infancy to childhood. There weren't any significant developmental changes of the negative deflection $N_{i320/c225}$ for the ERPs to the deviant stimuli.

Differences in amplitudes between the control and at-risk groups. No group differences were found for the amplitudes of the negative deflection $N_{i320/c225}$.

Developmental changes in latencies. For latencies of the negative deflection $N_{i320/c225}$, an Age × Stimulus type × Scalp site × Group MANOVA showed a significant Age main effect, $F(1, 34) = 125.06$, $p < .000$. Latencies shortened as a function of age (Appendix, Tables 7 and 8). There was a Stimulus type main effect, $F(1, 34) = 45.32$, $p < .000$, which indicated that the responses to the standard versus deviant stimuli differed from each other across both ages and groups. Besides, differences in responses to different stimulus types changed with age (Age × Stimulus type), $F(1, 34) = 143.47$, $p < .000$. This was caused by longer latencies of standard stimuli at the age of 6 months, and opposite responding between stimulus types at 6,5 years. A significant interaction Age × Scalp site, $F(7, 28) = 5.80$, $p < .000$, $\Lambda = .408$, showed that latencies changed differently at different scalp sites between ages. There was also a significant interaction Age × Stimulus type × Scalp site, Greenhouse-Geisser corrected ANOVA, $F(7, 238) = 3.25$, $p < .015$. This indicated that different stimulus types were responded differently in infancy and in childhood at different scalp sites.

Standard and deviant stimuli. No developmental changes were found in the separate analysis for the latencies of the standard stimuli . For the latencies of the deviant stimuli an additional analysis Age × Hemisphere × Anterior-posterior scalp site × Group revealed a significant Hemisphere main effect, $F(1, 34) = 4.46$, $p < .042$. Latencies at the right hemisphere were longer than latencies at the left hemisphere.

Differences in latencies between the control and at-risk groups. Any group differences weren't found in the analysis of Age × Stimulus type × Scalp site × Group MANOVA.

Standard and deviant stimuli. There weren't any group differences in the separate analysis for the latencies of the standard stimuli. For the ERPs to the deviant stimuli there was a significant interaction Hemisphere × Group, $F(1, 34) = 5.74$, $p < .022$. Groups differed also in their anterior-posterior scalp topography between the hemispheres (Hemisphere × Anterior-posterior × Group), $F(2, 33) = 3.97$, $p < .028$, $\Lambda = .805$. These interactions were a result of earlier latencies at the central and parietal channels at the right hemisphere than at the left hemisphere in infancy in the control group, while in the at-risk group the responses were opposite.

3.4 Negative deflection $N_{i450/c450}$ in the Long atta deviant condition

Developmental changes in amplitudes. Mean amplitude values of the negative deflection $N_{i450/c450}$ were analysed with an Age × Stimulus type × Scalp site × Group MANOVA. This revealed a significant Age main effect, $F(1, 34) = 11.03$, $p < .002$. This was due to more negative amplitudes in childhood than in infancy (Pictures 2a and 2b; Appendix, Tables 9 and 10). There was also a significant interaction Age × Scalp site, $F(7, 28) = 5.71$, $p < .000$, $\Lambda = .411$, which indicated that the scalp distribution changed as a function of age. Scalp site changes between age-groups led to more detailed analyses of Age × Hemisphere × Anterior-posterior scalp site × Group for both of the stimulus types separately.

Standard and deviant stimuli. For the ERPs to the standard stimuli there was a significant interaction Hemisphere × Anterior-posterior scalp site, $F(2, 33) = 3.39$, $p < .046$, $\Lambda = .829$, as well as an Age × Hemisphere × Anterior-posterior interaction, $F(2, 33) = 3.61$, $p < .038$, $\Lambda = .820$. Amplitude means showed that the left hemisphere was more negative at the age of 6 months. Until the age of 6,5 years differences between the hemispheres changed to opposite direction but parietal electrodes stayed more negative at the left hemisphere. There weren't any significant developmental changes for the ERPs to the deviant stimuli.

Differences in amplitudes between the control and at-risk groups. The control and at-risk groups differed in their responses to different stimulus types (Stimulus type × Group), $F(1, 34) = 5.26$, $p < .028$. In infancy, a Stimulus type × Scalp site × Group MANOVA gave a significant interaction Stimulus type × Group at the left hemisphere, $F(1, 34) = 4.75$, $p < .036$. In childhood the groups differed in their responses to different stimulus types at all the scalp sites, $F(1, 34) = 4.52$, $p < .041$. These differences between the groups were a result of more negative responses to deviant stimuli in the control group, while in the at-risk group responses were more usually larger to standard stimuli. There was also a Stimulus type × Scalp site × Group interaction, $F(7, 28) = 3.04$, $p < .016$, $\Lambda = .567$, which indicated that the groups differed in their responses to different stimulus types at different scalp sites.

Standard and deviant stimuli. No group differences were found for the ERPs to the standard and deviant stimuli separately.

Developmental changes in latencies. An Age × Stimulus type × Scalp site × Group analysis for peak latencies revealed a significant Stimulus type main effect, $F(1, 34) = 765.14$, $p < .000$, which was due to a longer silence gap in the deviant stimulus long *atta* (Appendix, Tables 11 and 12).

Standard and deviant stimuli. There weren't any developmental changes in the latencies of the negative deflection $N_{i450/c450}$ to the standard stimuli. For the ERPs to the deviant stimuli there was an Age main effect, $F(1, 34) = 4.32$, $p < .045$, indicating that the latencies of the deviant stimuli were longer at 6,5 years than at 6 months.

Differences in latencies between the control and at-risk groups. There were developmental differences in the latencies between the groups at different scalp sites (Age × Scalp site × Group), $F(7, 28) = 3.22$, $p < .012$, $\Lambda = .553$.

Standard and deviant stimuli. For the ERPs to the standard stimuli there weren't any group differences. For the latencies of the response to the deviant stimuli there was a significant interaction Hemisphere × Anterior-posterior × Group, $F(2, 33) = 3.50$, $p < .042$, $\Lambda = .82$, indicating that groups responded differently at different scalp sites between the hemispheres. In addition, these differences changed with age in a complicated way (Age × Hemisphere × Anterior-posterior × Group, $F(2, 33) = 6.35$, $p < .005$, $\Lambda = .722$. In infancy, the latencies at the frontal and parietal electrodes were longer at left than at the right hemisphere in the control group, and these responses were opposite in the at-risk group. In childhood, latencies at the frontal and parietal electrodes were shorter at left than at the right hemisphere in the control group, and in the at-risk group only the latencies at the frontal electrodes were shorter at left than at the right hemisphere.

3.5 Waveforms in the Short ata deviant condition

In the Short ata deviant condition, the probabilities of the short *ata* and the long *atta* were reversed. The probability of the short *ata* was now 10 % and 80 % for the long *atta*. The purpose was to further test whether the stimuli as such or their difference determined ERP and related group differences. If the stimulus context had no effect to the ERP waveform, the brain's response to the short *ata* as the deviant stimulus should be similar to that elicited by the same *ata* as a frequently occurring standard stimulus (in the Long atta deviant condition).

Results considering the Short ata deviant condition in infancy were reported by Leppänen et al. (2002). Their main findings are displayed in the Figure 3a. In infancy in the control group the short *ata* as the deviant stimulus elicited an additional negative peak at 550 ms compared to the short *ata* as the standard stimulus (Figure 3a). This peak occurred 380 ms after the deviant and standard stimulus difference. In the at-risk group (Figure 3b), it was completely absent at both the hemispheres. At the latency range of this negative deflection Leppänen et al. (2002) found a Group main effect, which indicated that responses differed between the groups.
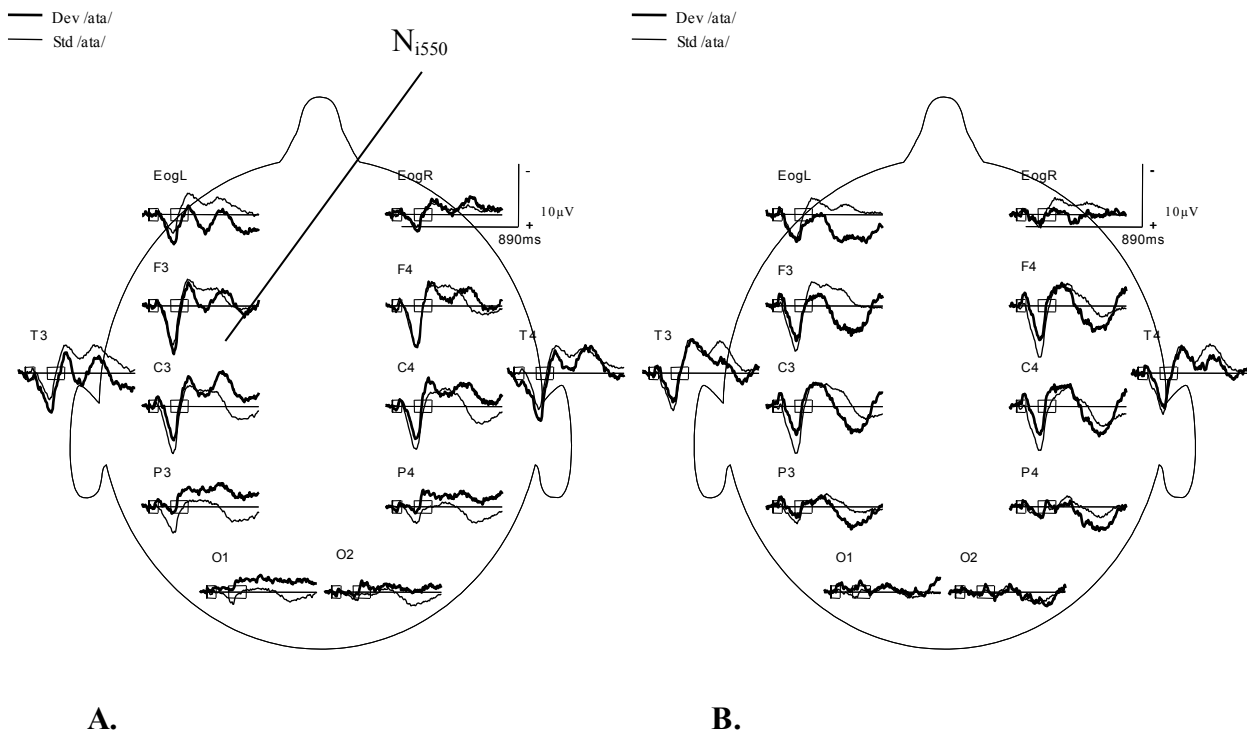
FIGURE 3. ERPs (averaged across infants) at the age of 6 months in the Long atta deviant condition vs. in the Short ata deviant condition. **(A)** Responses to short *ata* as deviant (probability 10%) vs. short *ata* as standard (probability 80%) in the control group (n = 12 and 19 for the Short ata and Long atta deviant conditions, respectively). There exists an additional negativity at approximately 550 ms in the deviant *ata* condition. That peak reflects the change detection. **(B)** Responses to deviant *ata* vs. standard *ata* in the at-risk group (n = 12 and 17 for the Short ata and Long atta deviant conditions, respectively). The second negative deflection is absent in the at-risk group, which means defective processing of the deviant stimulus. Stimuli are represented with small boxes on the calibration line. Negativity up.

At the age of 6,5 years in the control group (Figure 4a) the response to the deviant short *ata* was more negative than that to the standard short *ata*. Negative peaks N225, N310 and N450 were more fused together and the largest negative peak occurred at about 450 ms (270 ms after the occurrence of the deviant and standard stimulus difference). In the at-risk group (Figure 4b) the deviant short *ata* elicited more similar waveform compared to the standard short *ata* than in the control group. Negative peak N310 was the only peak that seemed to be more negative for the deviant short *ata*. (Tables 13 and 14).
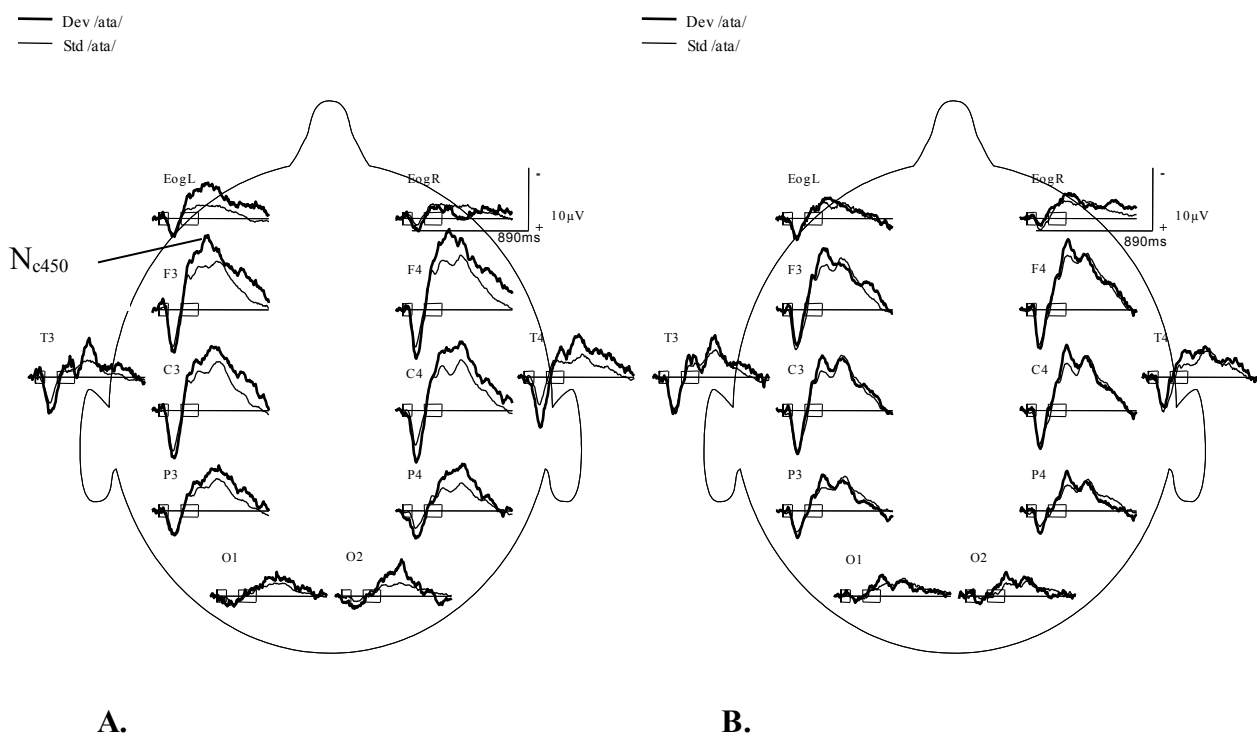
FIGURE 4. ERPs (averaged across children) at the age of 6,5 years in the Long atta deviant condition vs. in the Short ata deviant condition. **(A)** Responses to short *ata* as deviant (probability 10%) vs. short *ata* as standard (probability 80%) in the control group (n = 7 and 19 for the Short ata and Long atta deviant conditions, respectively). The waveform is more negative for the deviant *ata* than for the standard *ata*. **(B)** Responses to deviant *ata* vs. standard *ata* in the at-risk group (n = 9 and 17 for the Short ata and Long atta deviant conditions, respectively). The response to the deviant *ata* is only a bit more negative for the deflection N310 than the response to the standard *ata*. Stimuli are represented with small boxes on the calibration line. Negativity up.

3.6 Negative deflection N_{c450} in the Short ata deviant condition

Firstly, the responding differences between *ata* as standard and *ata* as deviant were analysed in both groups separately. A Scalp site × Stimulus type (*ata* as standard vs. *ata* as deviant) repeated measures MANOVA for the negative deflection N_{c450} gave a significant Stimulus type main effect in the control group, $F(1, 24) = 4.30$, $p < .049$. This indicated that in the control group the responding to *ata* as standard was different from that to *ata* as deviant. On the contrary, in the at-risk group there was no significant difference between the responses to *ata*s presented in different contexts, $F(1, 24) = .03$, $p > .05$.

Secondly, the responses between the groups were compared in the Short ata deviant condition. Because the groups were small (n = 7 and 9 in the control and in the at-risk groups, respectively), analyses were made separately for each electrode between the groups using Mann-Whitney U –tests. Only the negative deflection N_{c450} was compared between the groups. There was a significant difference between the groups at the C4-electrode at the right hemisphere, $Z = -2.170$, $p < .030$, Mann-Whitney U = 11.00. Responses at the other electrodes didn't differ between the groups.

4. DISCUSSION

4.1 Developmental changes in ERPs between 6 months and 6,5 years

<u>Positive deflection $P_{i190/c110}$.</u> The deflection $P_{i190/c110}$ could be thought to represent the adult P1 deflection, because the P1 has been observed to develop very early. Ceponiene et al. (2002) have suggested that the P1 reflects auditory sensory processes. Probably, also in this study the deflection $P_{i190/c110}$ represents an obligatory response to sounds in general. The amplitude and latency changes of the P1 were similar for the responses to the standard and deviant stimuli. Amplitudes of the first positive deflection $P_{i190/c110}$ decreased from infancy to childhood. For example, Kushnerenko et al. (2002) have found that the amplitude of P1 grew from birth to 3 months, but didn't grow anymore after that up to 12 months. Ponton et al. (2000) have shown amplitudes of P1 to decrease from the age of 5 years to adulthood. Thus, P1 amplitude seems to be at it's height in infancy, and then gets smaller as age increases. The diminution of the amplitudes of P1 as a function of age could indicate that the effort needed in sensory processes decreases as age increases.

The latencies of the $P_{i190/c110}$ had a strong correspondence with the latencies of the P1 in infancy and in childhood reported in another studies. Latencies of the $P_{i190/c110}$ shortened from 190 ms in infants to 110 ms in children. This shortening of the latencies indicates that the response speed to the physical features of sounds had become faster as age had increased. Kushnerenko et al. (2002) have reported a latency of 160 ms for the P1 at the age of 6 months. In childhood, latencies have been 80 – 90 ms (Ceponiene et al., 1997; Ponton et al., 2000; Sharma et al., 1997). Responses to the standard stimuli were more positive at the left hemisphere at both ages. However, in the study of Ceponiene et al. (2002) the P1 predominated around the vertex and in the study of Paetau et al. (1995) P1 responses didn't differ between the hemispheres.

<u>Negative deflection $N_{i320/c225}$.</u> The relationship of the deflection $N_{i320/c225}$ to mature adult ERP peaks is ambiguous. Probably, $N_{i320/c225}$ could be a precursor of the N2 peak, because N2 has been observed to appear to ERP waveform very early, much in the same

way as the first positive component P1. In the study of Kushnerenko et al. (2002) negative peak with the latency of 250 ms, peaking after the positive wave P150, was considered the correlate of adult N2 peak in infants under 12 months. Amplitudes of the negative deflection $N_{i320/c225}$ increased from 6 months to 6,5 years. In other studies N2 has been reported to dominate auditory ERPs in childhood until adolescence (Ponton et al., 2000). In the study of Ceponiene et al. (2002), N2 was the largest peak in the ERP waveform in childhood, but amplitudes decreased from 4 to 9 years. Thus, the maximum of the N2 peak amplitude is probably later than at the age of 6 months but earlier than at the age of 4 years.

Latencies of the deflection $N_{i320/c225}$ shortened from the latency of 320 ms in infancy to the latency of 225 ms in childhood, so the response speed of the $N_{i320/c225}$ became faster by age. These latencies of $N_{i320/c225}$ are quite consistent with the earlier reported latencies of N2. Latencies have been reported to be 250 ms in infancy (Kushnerenko et al., 2002), and 200 – 290 ms in children aged 4 – 9 years (Ceponiene et al., 1997 and 2000; Paetau et al., 1995; Ponton et al., 2000).

The responses of $N_{i320/c225}$ to the standard and deviant stimuli were different in both groups at both ages. In infancy both groups responded more negatively to the standard stimuli and in childhood more negatively to the deviant stimuli. Besides, latencies were longer for the standard than for the deviant stimuli in infancy, and this was contradictory in childhood. In infants the peak of the negative deflection $N_{i320/c225}$ occurred about 150 ms, and in children about 60 ms, after the standard and deviant stimuli started to differ in their structures. Consequently, different responding to the standard and deviant stimuli can't be interpreted as mismatch response, because $N_{i320/c225}$ appears too early compared to the latencies reported for MMN in earlier studies (for example Cheour et al., 1998; Kushnerenko et al., 2002b; Kraus et al., 1992). Kushnerenko et al. (2001) have reported of a duration specific N2 component. By extending the tone stimuli, N2 showed similar enlargement and longer latencies in infants and in adults compared to shorter stimuli. This is in line with our findings in childhood, when the deviant stimuli, which differed from the standard stimuli by its duration, were responded larger and later than the shorter standard stimuli. However, in infancy the responding was contrary. This could be due to the appearance of an

additional negative peak, and a positivity peaking before that to the deviant stimuli in infancy. In other words, in infancy N2 sustained longer to the standard stimuli because there wasn't anything new to respond. N2 response to the deviant stimuli was smaller because a positivity before an additional negative peak may have overlapped with it. Although different stimulus types were responded differently there wasn't any group differences for this negative deflection.

In childhood the deflection $N_{i320/c225}$ to the standard stimuli was only a small protuberance of the slope of the following negative peak N310. This small peak could probably present N1 peak instead of N2 in children. N1 peak has been observed to appear in the ERP waveform already at the age of 5 years when ISI has been long enough, at least 1200 ms (Paetau et al., 1995). This is due to a longer recovery period in children than in adults. N1 may also overlap with more robust deflections P1 and N2 and thus be invisible or very small. In the study of Ceponiene et al. (1998) with children aged 7 to 9 years, N1 started to emerge into the ERP waveform with an ISI of 1400 ms and longer, but a small knot which was fused together with the later negativity N250 appeared already with shorter ISIs of 350 ms and 700 ms. Karhu et al. (1997) showed that in children aged 9 years N1 was largest in response to the first tone in a sequence of four tones with ISI of 1 s. The second tone elicited significantly smaller amplitude, which was about half smaller than the first amplitude. Decreasing of N1 by repetition caused it to fuse with the N2 peak. Thus, in this study the negative deflection $N_{i320/c225}$ could also be thought as a precursor of the adult mature N1 peak.

4.2 Differences between the control and at-risk groups in infancy and in childhood

Positive deflection $P_{i190/c110.}$ Groups differed in infancy in their positive, obligatory deflection $P_{i190/c110}$. Responses in the at-risk group were more positive than in the control group to the deviant than to the standard stimuli. Leppänen et al. (2002) have found differences between the groups in infancy also in the positive response P470, which wasn't analysed in the present study. The at-risk infants responded more positively than the controls and this was interpreted as higher obligatory responsiveness to sound onsets

before MMN-like response. Larger obligatory responsiveness to the deviant stimuli in the at-risk infants could indicate that dyslexia is linked with an abnormal sensitivity to sounds in general at least in infancy. Leppänen, Pihko, Eklund, & Lyytinen (1999) have found more positive responses to vowel duration change in newborns at-risk for dyslexia at the latency range of 280 – 500 ms.

However, differences between the groups regarding this obligatory positive deflection disappeared in childhood, because the amplitudes for the standard stimuli changed to more positive direction in the at-risk group as age increased. The children at-risk for dyslexia weren't more sensitive to sounds in childhood any more and considering this the groups had become closer to each other. These results in childhood were inconsistent with the results of Maurer et al. (2003b) who found that the children aged 6 to 7 years at-risk for dyslexia responded more positively than the control children at the latency range of 109 – 140 ms. This difference between groups was more clear to phoneme than frequency deviants, thus it could partly explain why the at-risk children in the present study didn't respond more positively to the deviant stimuli, which differed in its temporal structure from the standard stimuli.

Negative deflection $N_{i450/c450}$ in the Long atta deviant condition. In infancy, there was an additional late negative peak at the latency of 600 ms for the deviant long *atta* compared to the waveform elicited by the standard short *ata*. In the study of Leppänen et al. (2002) the late negative deflection N600 to the deviant stimuli was larger in the control than in the at-risk group at the left hemisphere. In the present study, with smaller group sizes these group differences to the deviant stimuli weren't found in infancy, but instead the groups differed in their responses between the standard and deviant stimuli at the left hemisphere. Leppänen et al. (2002) proposed that this additional negative deflection to the deviant stimuli represents at least partly the change detection response in infancy.

The last negative peak changed a lot with age, which indicates a bit different change detection processes in infancy and in childhood. In childhood there wasn't an additional negative peak to the deviant stimuli any more, but the ERP waveform was similar for the standard and deviant stimuli. However, in the control group the responses to the deviant stimuli were more negative than to the standard stimuli. This negativity

probably represents change detection or MMN response, which usually appears in an oddball paradigm used in the present study. The late negative deflection to the deviant stimuli resembled probably also obligatory responses to the second part /ta/ of the deviant stimuli *atta* than merely the MMN. In childhood the groups differed in their responses between the standard short *ata* and the deviant long *atta* at all the scalp sites. In the control group the deviant stimuli were responded more negatively than the standard stimuli while in the at-risk group this was opposite. The present study shows that groups still differed in childhood.

Negative deflection N_{c450} in the Short ata deviant condition. Responses to the same stimulus in different contexts differed in infancy between the groups (Leppänen et al., 2002). The deviant stimuli *ata* elicited an additional negative peak compared to the waveform elicited by the *ata* as standard. However, the at-risk infants responded similarly to the same stimulus, regardless of the stimulus context. The additional negative deflection was thought to represent the MMN, because the appearance of this peak was independent of attention, and the latency (380 ms) was in line with the reported infant MMN latency (Cheour et al., 1998b).

When same stimuli were presented in different contexts in childhood, groups still differed in their responses. The change detection response in childhood was different from that in infancy as was in the Long atta deviant condition. The waveform was significantly more negative in the control group for the *ata* as deviant than for the *ata* as standard. The at-risk children responded more to the physical features of the stimuli than compared it to the context in which it was presented. Groups differed also in their responses to the deviant *ata* at the central electrode at the right hemisphere. In infancy, Leppänen et al. (2002) found differences on the whole scalp, so the location of this processing difference between groups changed from infancy to childhood. One cause for the fact that differences between groups were found only at one channel in childhood may be found from the smaller group sizes in childhood (in the study of Leppänen et al. (2002) n = 12 and 12, while in the present study n = 7 and 9 in the control and the at-risk groups, respectively).

The peak latency of the negative deflection N_{c450} was about 270 ms after the difference of the standard and deviant stimuli. This is consistent with the studies of Kraus et al. (1992, 1993 and 1995), who have reported the latency of MMN in children to be about 220–230 ms. As well as in infancy, in childhood this negative peak could be interpreted as MMN. Stimuli differed by their temporal features, thus the infants and children at-risk for dyslexia have a deficient passive discrimination ability of temporal features of sounds. This supports the view that dyslexia is linked with problems in auditory processing (Rey et al., 2002; Tallal, 1980).

Permanence of the group differences from infancy to preschool age. Groups differed at both ages in the temporal processing of speech, but the more sensitive responsiveness of the at-risk infants to sounds per se found in infancy had disappeared in childhood. Thus differences between the groups had become a bit smaller in childhood. There weren't found any new group differences in childhood.

Smaller group sizes in the present study compared to the study of Leppänen et al. (2002) might be one cause for the smaller group differences in childhood. Another cause for the alleviated differences between groups might be the increased influence of environment in childhood. Developmental dyslexia has been found to have a genetic ground (Pennington, 1995; Taipale et al., 2003). In infancy the influence of genes to brain function and behaviour is remarkable, but when a child gets older the environmental factors play more important role. Molfese, Molfese, Key, & Kelly (2003) have reported that the amount of stimulation for language and reading in home is related to discrimination ability of speech and non-speech measured by ERPs. Consequently, if a child in at-risk for developmental dyslexia gets fruitful experiences and enough support in home environment, the probability of manifestation of language learning deficits could become smaller.

Bishop & McArthur (2004) have found a proof that SLI and perhaps also dyslexia may be due to a delayed maturation of auditory processing. In their study the ERPs to tone pairs of older participants with SLI differed from the ERPs of age-matched controls, but resembled ERPs of younger controls. Considering this, it might be thought that in the present study the alleviated differences between the groups at the age of 6,5 years might

partly be caused by the fact that the at-risk group has slowly caught up this maturation delay of auditory processing.

It has been found that ERPs recorded at birth have discriminated infants who have later been categorised as dyslexic, poor or normal readers (Molfese, 2000). In the future, it would be interesting to find out if ERP peaks could be used as a tool in individual diagnostics for language learning deficits. The individual ERPs of those children of the at-risk group who later turn out to be dyslexic could be examined closer and find out if there is enough reliability in individual ERPs to predict later manifestation of dyslexia. This would enable to start individual rehabilitation earlier than it has been possible before.

Conclusions. Firstly, the present study investigated the developmental time courses of the ERP peaks. It was found that the latencies of the deflections $P_{i190/c110}$ and $N_{i320/c225}$ shortened with age. This indicates that responses become faster with age. Amplitudes decreased for the $P_{i190/c110}$ and this shows that the processing of physical features of sounds doesn't need as much effort in childhood as in infancy. The $P_{i190/c110}$ was thought to be a precursor of the adult P1 component. Amplitudes of the $N_{i320/c225}$ increased as a function of age and the $N_{i320/c225}$ was thought to represent the adult N2 component. In childhood there probably was some sign of N1. Secondly, the differences in the automatic detection of temporal changes in speech were studied between the control group and the at-risk group for dyslexia. The groups differed at the both ages in their automatic detection of temporal changes of speech. This shows that individuals at-risk for developmental dyslexia have deficient perception of temporal features of speech and results support the view that dyslexia is linked with deficits in auditory processing. However, the more sensitive obligatory responsiveness in the at-risk group found in infancy had disappeared in childhood, so deficits in speech perception had somewhat eased from infancy to childhood.

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APPENDIX

TABLE 1. Mean peak amplitudes in microvolts (μV) for the deflection $P_{i190/c110}$, standard short *ata*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	6,3	3,3	7,3	2,3
C3	7,5	3,1	7,3	2,4
T3	4,2	2,7	5,8	2,9
P3	4,1	2,0	2,6	1,8
F4	6,5	3,4	8,1	3,3
C4	7,1	3,1	7,1	3,1
T4	5,7	3,7	6,4	2,7
P4	2,9	1,9	2,3	1,8
Childhood				
F3	6,2	2,2	6,5	3,0
C3	6,5	2,1	6,9	2,6
T3	4,2	1,8	5,3	2,1
P3	3,9	1,5	3,5	1,8
F4	6,0	2,1	5,8	3,1
C4	5,8	1,9	5,7	3,0
T4	4,4	2,0	5,7	2,8
P4	3,2	1,6	2,8	1,9

TABLE 2. Mean peak amplitudes in microvolts (μV) for the deflection $P_{i190/c110}$, deviant long *atta*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	5,4	4,0	8,2	3,0
C3	6,5	3,2	8,5	3,8
T3	4,4	3,2	7,6	4,0
P3	2,5	2,6	4,5	4,0
F4	5,4	4,6	9,0	4,1
C4	5,8	4,6	8,3	3,8
T4	4,8	3,7	7,5	2,7
P4	2,6	2,5	3,7	3,0
Childhood				
F3	6,3	3,0	6,1	2,7
C3	5,8	2,4	6,7	2,8
T3	3,0	1,7	5,0	2,2
P3	3,5	1,8	4,0	2,1
F4	6,3	3,8	5,3	2,3
C4	5,6	3,1	5,7	2,7
T4	3,9	1,9	5,2	2,7
P4	3,0	2,3	3,3	1,8

TABLE 3. Peak latencies in milliseconds (ms) for the deflection $P_{i190/c110}$, standard short *ata*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	185,8	10,3	178,2	13,2
C3	186,6	10,9	172,1	21,2
T3	177,1	35,6	183,2	14,5
P3	187,4	19,4	171,8	45,3
F4	176,8	16,6	176,2	13,6
C4	178,9	15,8	175,9	21,4
T4	189,2	14,0	177,9	29,1
P4	195,3	51,7	189,7	60,4
Childhood				
F3	108,2	9,9	102,6	15,9
C3	111,1	17,1	112,1	25,7
T3	119,5	21,9	115,9	25,8
P3	112,9	20,2	115,0	25,3
F4	102,4	11,2	102,4	15,5
C4	107,9	15,8	103,2	15,3
T4	114,2	12,7	117,6	18,8
P4	107,9	18,1	106,5	33,2

TABLE 4. Peak latencies in milliseconds (ms) for the deflection $P_{i190/c110}$, deviant long *atta*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	177,4	35,8	179,4	24,6
C3	172,1	25,0	182,4	24,6
T3	164,2	40,1	189,7	19,8
P3	175,5	41,1	217,9	75,0
F4	181,3	31,5	171,5	22,5
C4	177,6	23,4	170,9	23,2
T4	164,7	49,1	178,2	32,6
P4	176,3	69,5	169,4	52,9
Childhood				
F3	106,6	15,0	103,2	12,6
C3	115,3	19,1	111,8	25,3
T3	112,9	38,7	111,2	27,5
P3	114,7	22,7	125,3	35,7
F4	104,7	12,0	95,9	11,4
C4	103,9	16,1	94,7	9,9
T4	112,9	13,9	120,6	25,2
P4	110,3	30,8	113,2	43,7

TABLE 5. Mean peak amplitudes in microvolts (μV) for the deflection $N_{i320/c225}$, standard short *ata*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	-5,1	3,1	-4,3	3,5
C3	-4,2	4,0	-4,2	3,6
T3	-4,9	3,4	-5,5	3,4
P3	-1,6	2,8	-1,8	2,2
F4	-4,6	3,0	-3,4	3,8
C4	-3,7	3,1	-2,8	3,0
T4	-4,9	3,0	-4,3	3,1
P4	-1,3	2,5	-1,6	1,5
Childhood				
F3	-5,6	4,0	-5,2	4,6
C3	-4,8	3,4	-4,3	4,4
T3	-1,6	2,5	-1,9	3,1
P3	-2,6	2,3	-2,3	3,0
F4	-6,2	3,8	-5,4	4,2
C4	-5,4	3,0	-4,7	3,9
T4	-2,8	2,5	-1,8	3,6
P4	-2,8	2,2	-2,0	2,8

TABLE 6. Mean peak amplitudes in microvolts (μV) for the deflection $N_{i320/c225}$, deviant long *atta*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	-4,1	4,1	-3,2	3,8
C3	-3,9	4,0	-2,3	3,6
T3	-4,2	3,7	-3,1	3,6
P3	-2,2	3,7	-,7	3,1
F4	-4,5	5,4	-2,8	4,2
C4	-4,3	5,5	-1,7	3,3
T4	-5,0	4,1	-3,5	3,3
P4	-2,4	2,9	-1,0	2,7
Childhood				
F3	-9,9	5,1	-9,6	4,4
C3	-9,6	5,0	-8,6	3,4
T3	-4,7	4,0	-4,5	2,8
P3	-5,7	3,5	-5,4	2,8
F4	-10,6	5,0	-10,5	4,3
C4	-9,9	4,0	-9,1	3,9
T4	-5,3	4,1	-5,2	3,7
P4	-5,6	2,9	-5,5	3,3

TABLE 7. Peak latencies in milliseconds (ms) for the deflection $N_{i320/c225}$, standard short *ata*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	310,0	35,0	309,7	29,4
C3	312,6	39,9	317,9	35,4
T3	322,6	27,0	318,5	24,0
P3	312,9	43,6	299,7	49,0
F4	310,8	37,6	317,1	36,3
C4	315,0	38,7	332,9	30,6
T4	317,6	27,1	321,8	18,4
P4	294,2	52,9	311,5	57,2
Childhood				
F3	226,6	11,8	231,5	11,0
C3	230,3	12,9	235,9	7,5
T3	207,6	33,6	226,2	18,5
P3	226,8	22,6	230,0	27,4
F4	224,7	23,5	230,0	12,4
C4	231,1	11,6	235,9	8,3
T4	228,2	15,6	230,6	12,1
P4	222,4	29,0	232,6	11,6

TABLE 8. Peak latencies in milliseconds (ms) for the deflection $N_{i320/c225}$, deviant long *atta*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	305,3	43,6	318,2	35,2
C3	316,1	36,6	307,4	31,4
T3	320,3	23,8	319,1	15,3
P3	303,9	38,8	280,9	58,5
F4	308,2	42,6	316,3	27,0
C4	301,3	42,3	315,3	36,3
T4	316,8	31,8	327,9	19,8
P4	278,2	56,7	304,1	49,8
Childhood				
F3	285,5	49,3	300,0	50,6
C3	283,2	48,9	289,1	41,1
T3	260,0	67,9	272,4	61,7
P3	292,6	48,1	312,9	60,4
F4	297,4	44,3	300,3	38,4
C4	299,7	42,6	304,7	47,7
T4	266,8	47,1	285,9	59,4
P4	297,4	48,2	339,1	50,7

TABLE 9. Mean peak amplitudes in microvolts (μV) for the deflection $N_{i450/c450}$, Standard short *ata*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	-5,0	3,1	-5,4	3,0
C3	-4,7	3,8	-5,2	2,9
T3	-5,6	3,0	-6,5	3,6
P3	-2,2	2,9	-2,6	1,8
F4	-4,7	3,3	-4,9	3,0
C4	-4,2	3,2	-4,1	2,7
T4	-5,6	2,8	-5,6	4,6
P4	-1,5	2,4	-2,2	1,8
Childhood				
F3	-8,5	4,2	-8,7	3,2
C3	-8,3	3,8	-8,9	2,9
T3	-3,4	3,5	-4,6	2,8
P3	-5,5	2,7	-5,5	2,0
F4	-8,9	4,3	-9,5	2,9
C4	-8,2	3,9	-9,0	2,6
T4	-3,9	3,6	-4,8	2,4
P4	-4,7	2,4	-5,1	2,2

TABLE 10. Mean peak amplitudes in microvolts (μV) for the deflection $N_{i450/c450}$, deviant long *atta*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	-6,1	3,7	-5,3	4,4
C3	-6,6	3,7	-4,0	3,6
T3	-5,4	3,4	-4,4	4,5
P3	-4,5	3,6	-2,9	2,9
F4	-6,7	5,1	-5,9	5,0
C4	-6,8	5,4	-4,7	4,3
T4	-5,1	3,5	-5,3	4,5
P4	-4,4	3,5	-3,8	4,0
Childhood				
F3	-8,3	4,0	-7,5	3,1
C3	-9,0	3,8	-7,5	2,4
T3	-4,8	3,0	-4,7	3,2
P3	-6,1	2,4	-4,3	1,9
F4	-8,7	3,8	-8,8	4,0
C4	-8,5	3,3	-7,5	3,4
T4	-4,4	3,5	-4,4	3,8
P4	-5,3	2,4	-4,4	2,3

TABLE 11. Peak latencies in milliseconds (ms) for the deflection $N_{i450/c450}$, standard short *ata*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	462,1	72,2	440,6	78,6
C3	459,5	74,5	434,4	70,1
T3	482,4	86,1	457,6	92,8
P3	433,4	63,9	417,4	54,6
F4	432,1	64,9	443,8	81,8
C4	443,2	68,3	415,9	63,4
T4	473,9	77,4	496,5	91,2
P4	438,4	69,1	426,5	67,5
Childhood				
F3	434,7	31,7	456,2	25,5
C3	447,4	31,8	457,9	23,0
T3	410,3	57,3	421,2	38,1
P3	446,3	33,7	472,1	24,9
F4	438,9	29,5	447,6	33,3
C4	453,4	24,7	450,3	21,2
T4	419,2	66,1	449,7	63,9
P4	451,6	29,4	458,5	20,1

TABLE 12. Peak latencies in milliseconds (ms) for the deflection $N_{i450/c450}$, deviant long *atta*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Infancy				
F3	600,8	64,8	605,3	49,4
C3	605,8	71,1	627,4	59,0
T3	638,9	67,1	645,6	68,2
P3	617,9	76,3	586,8	84,2
F4	594,5	57,9	614,4	57,4
C4	610,3	68,0	603,2	57,0
T4	631,8	63,9	635,3	42,0
P4	598,2	87,9	603,2	71,8
Childhood				
F3	614,5	29,4	622,9	36,4
C3	623,2	31,1	624,1	35,8
T3	573,7	55,5	570,0	45,1
P3	625,3	36,6	631,5	33,2
F4	623,2	31,1	630,3	34,0
C4	619,2	32,2	619,7	29,7
T4	597,1	60,0	591,2	82,1
P4	627,1	38,4	630,6	37,4

TABLE 13. Mean peak amplitudes in microvolts (µV) for the deflection (N_{c450}), deviant short *ata*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Childhood				
F3	-11,2	5,7	-8,2	3,8
C3	-10,9	4,3	-8,8	3,3
T3	-6,4	4,3	-6,3	2,1
P3	-7,4	2,8	-4,9	2,3
F4	-12,3	5,4	-9,4	4,4
C4	-11,2	3,8	-9,1	2,3
T4	-7,5	4,2	-5,5	3,7
P4	-7,7	2,6	-5,1	1,8

TABLE 14. Peak latencies in milliseconds (ms) for the deflection N_{c450}, deviant short *ata*

Electrode Site	Control group		Risk group	
	M	Sd	M	Sd
Childhood				
F3	457,9	53,3	450,6	33,4
C3	458,6	39,9	447,2	24,8
T3	406,4	74,0	418,3	11,7
P3	458,6	44,4	461,7	27,3
F4	461,4	49,9	452,8	36,7
C4	462,9	31,9	455,0	23,2
T4	406,4	72,6	409,4	62,1
P4	458,6	19,1	460,6	30,7